**U.S. Department of Labor**

**Office of Administrative Law Judges**
**525 Vine Street - Suite 900**
**Cincinnati, Ohio 45202**

**(513) 684-3252**
**(513) 684-6108 (FAX)**

**Issue date: 30Jul2001**

Case No: 2000-BLA-0768

In the Matter of

LARRY TAYLOR
Claimant

v.

SHARONDALE CORPORATION,
LOFTIS COAL COMPANY
Employers

KENTUCKY COAL PRODUCERS SELF-INSURANCE FUND,
OLD REPUBLIC INSURANCE COMPANY
Carriers

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

APPEARANCES:

Cynthia Mulliken, Esq.
For the claimant

David H. Neeley, Esq.
For the employer Sharondale

John T. Chafin, Esq.
For the employer Loftis Coal
and carrier Old Republic

Donna E. Sonner, Esq.
Office of the Solicitor
For the Director

BEFORE: JOSEPH E. KANE
Administrative Law Judge

**DECISION AND ORDER - DISMISSING LOFTIS COAL AND REMANDING CLAIM TO OWCP**

This proceeding arises from a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, 30 U.S.C. § 901 *et seq.* (the Act). Benefits are awarded to coal miners who are totally disabled due to pneumoconiosis. Surviving dependents of coal miners whose deaths were caused by pneumoconiosis may also recover benefits. Pneumoconiosis, commonly known as black lung, is a chronic dust disease of the lungs arising from coal mine employment. 20 C.F.R. § 718.201 (1996).

On May 17, 2000, this case was referred to the Office of Administrative Law Judges for a formal hearing. Following proper notice to all parties, a hearing was held on November 14, 2000 in Pikeville, Kentucky. The Director's exhibits were admitted into evidence pursuant to 20 C.F.R. § 725.456, and the parties had full opportunity to submit additional evidence and to present closing arguments or post-hearing briefs.

By Order dated March 6, 2001, the parties were ordered to file briefs by March 16, 2001 stating with specificity how application of the amended regulatory provisions will affect the outcome of this case. (March 6, 2001 Order). The claimant did not respond. Following review of the Director's and the employer's positions, I issued an Order on May 15, 2001 wherein I found that the amended regulations will not affect the outcome of the case, "as the changed subsections which could impact the case are either codifications of existing case law, inapplicable or no evidence has been submitted which would trigger that particular amended regulation." I therefore ruled that adjudication of the claim may proceed. (June 7, 2001 Order).

The Findings of Fact and Conclusions of Law that follow are based upon my analysis of the entire record, arguments of the parties, and the applicable regulations, statutes, and case law. They also are based upon my observation of the demeanor of the witness who testified at the hearing. Although perhaps not specifically mentioned in this decision, each exhibit and argument of the parties has been carefully reviewed and thoughtfully considered. While the contents of certain medical evidence may appear inconsistent with the

conclusions reached herein, the appraisal of such evidence has been conducted in conformance with the quality standards of the regulations.

The Act's implementing regulations are located in Title 20 of the Code of Federal Regulations, and section numbers cited in this decision exclusively pertain to that title. References to DX and EX refer to the exhibits of the Director and the employer, respectively. The transcript of the hearing is cited as "Tr." and by page number.

## ISSUES

The following issues remain for resolution:

1. whether either named employer is the responsible operator;

2. whether the miner has pneumoconiosis as defined by the Act and regulations;

3. whether the miner's pneumoconiosis, if any, arose out of coal mine employment;

4. whether the miner is totally disabled;

5. whether the miner's disability is due to pneumoconiosis;

6. whether the evidence establishes a change in conditions or a mistake in a determination of fact within the meaning of §725.310; and

7. whether the evidence establishes a material change in condition, within the meaning of § 725.309(d), since the denial of the previous claim.

(Tr. 8-13; DX 180). As a review of the record shows that a request for modification is before me, and that a duplicate claim subject to § 725.309(d) was never filed in this case, I find that issue number 7 is not applicable.

## FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and Procedural History

The claimant, (Dallas) Larry Taylor, was 57 years old at the time of the hearing and has an eighth grade education. He has one dependent, his wife, for purposes of augmentation of benefits. (DX 1; Tr. 29-31). The parties stipulated that the claimant had at least 20.5 years of coal mine employment. (Tr. 9-10, 17). He last worked in August 1985. (Tr. 22).

The claimant testified that he began smoking cigarettes around the age of seventeen or eighteen. In the past year and a half, he had cut back his smoking to approximately ten cigarettes per day. (Tr. 32).

The claimant filed a claim for benefits under the Act on January 30, 1986. (DX 1). A formal hearing was held before a previous administrative law judge on June 6, 1988, at which time counsel for Loftis Coal Company appeared and defended the claim. (DX 77). By Decision and Order dated December 15, 1989, the administrative law judge dismissed Loftis Coal Company from the case, finding that Sharondale Coal Company should have been named as the responsible operator. Therefore, the judge held that the Black Lung Disability Trust Fund would be liable for the payment of any benefits. As to the merits of the claim, he found that the claimant did not establish that he had pneumoconiosis or other pulmonary condition caused by his coal mine employment (DX 84). The claimant appealed that denial to the Benefits Review Board ("the Board"). On September 28, 1992, the Board issued its Decision and Order affirming in part, vacating in part, and remanding the claim for further consideration consistent with its opinion, specifically Dr. Penman's diagnosis of coal workers' pneumoconiosis. (DX 100).

On remand, the judge again denied benefits on March 31, 1993, finding that Dr. Penman's opinion was not well-documented and thus entitled to little weight. (DX 102). The claimant again appealed to the Board, and on March 30, 1995, the Board again remanded the case. The Board affirmed the findings surrounding the physicians' opinions, but remanded for the administrative law judge to reconsider the evidence in light of the holding of the United States Court of Appeals for the Sixth Circuit in *Woodward v. Director, OWCP*, 991 F.2d 314, 321 (6th Cir. 1993), decided in the interim. (DX 118).

On the second remand, the claim was again denied by the administrative law judge on July 26, 1995 for failure to establish pneumoconiosis. The issue of total disability was not reached. (DX 119). The Board affirmed that denial on October 18, 1996 (DX 134), as did the United States Court of Appeals for the Sixth Circuit on September 12, 1997. (DX 136).

The claimant timely filed a request for modification on October 20, 1997. (DX 137). The District Director, Office of Workers' Compensation Programs ("OWCP") denied the request on April 3, 1998, October 1, 1998, and March 11, 1999. (DX 149, 164, 171). The claim was then forwarded to the Office of

Administrative Law Judges ("OALJ") on June 18, 1999 with the District Director as the responding party (DX 177), but was

remanded at the request of the District Director for the naming of Sharondale Corporation as the responsible operator. (DX 178). Subsequent to that remand, both Sharondale and Loftis were named and notified of the request for modification. Both controverted based on both their liability and the claimant's eligibility, with Sharondale citing to *Crabtree v. Bethlehem Steel Corporation*, 7 BLR 1-354 (1984) on the issue of liability. The OWCP denied the request for modification again on January 27, 2000. (DX 178). The claimant once again timely requested a formal hearing, and the case was referred to the OALJ on May 17, 2000. (DX 178, 180).

Responsible Operator

The previous judge held in his December 15, 1989 Decision and Order that Loftis Coal was not the responsible operator for this claim as the claimant had subsequently been employed by Sharondale for more than one year. The judge rejected the Director's argument that Sharondale was a successor operator to Loftis, an argument supported only by the testimony of the claimant, who had no business expertise. Therefore Loftis Coal was dismissed as a party.[1] Since Sharondale had not been named as a party, the judge found that any benefits would be payable by the Black Lung Disability Trust Fund. (DX 84). The Director did not appeal these findings. (DX 99, 125).

Section 725.310 states that:

> (a) Upon his or her own initiative, or upon the request of any party on grounds of a change in conditions or because of a mistake in a determination of fact, the [District Director] may, at any time before one year from the date of the last payment of benefits, or at any time before one year after the denial of the claim, reconsider the terms of an award or denial of benefits.
>
> ....

[1] Additionally, 20 C.F.R. § 725.493(a)(3)(iii) provides that the successor operator is the responsible operator, not the previous operator.

> (d) An order issued following the conclusion of modification proceedings may terminate, continue, reinstate, increase or decrease benefit payments or award benefits. Such order shall not affect any benefits previously paid, except that an order increasing or decreasing the amount of benefits payable may be made effective on the date from which benefits were determined payable by the terms of an earlier award. In the case of an award which is decreased, any payment made in excess of the decreased rate shall be subject to collection or offset under subpart G of this part.

20 C.F.R. § 725.310(a), (d).

The Director argues that the language of § 725.310 is broad and can revisit any issue of fact. Pertinently, the Sixth Circuit has held that modification may be relied upon by the District Director to correct misidentification in the case of the responsible carrier even where a final compensation order has been issued against the operator. *Caudill Construction Co. v. Abner*, 878 F.2d 179 (6th Cir. 1987). See also *USX Corp. v. Director, OWCP*, 978 F.2d 656 (11th Cir. 1992) (where the District Director erroneously transferred liability from the employer to the Trust Fund, the Director can request modification to transfer liability back to the employer if the request is timely made, that is within one year of the employer's last payment).

Also to be considered here is the "law of the case" doctrine. Black's Law Dictionary (5th ed., West Publishing Co., 1983) defines "law of the case" as:

> [T]he principle that if an appellate court has passed on a legal question and remanded the cause to the court below for further proceedings, the legal question thus determined by the appellate court will not be differently determined on a subsequent appeal in the same case where the facts remain the same.

Id. at p. 459. Here, the doctrine would operate to bar the Director from relitigating the issue of responsible operator,

as in not appealing the administrative law judge's findings before the Board, it had accepted them.[2] The facts have not changed.

However, the Sixth Circuit has held that departure from the doctrine is appropriate where the prior holding is "clearly erroneous" and its continued application would constitute a "manifest injustice." *Cale v. Johnson*, 861 F.2d 943, 947 (6th Cir. 1988)(citing to *Arizona v. California*, 460 U.S. 605 (1983)).

I find no error in the previous finding that Loftis Coal is not the responsible operator. Shardondale is clearly the last coal mine employer to employ the claimant for at least one cumulative year. The record does not contain any evidence that Sharondale is incapable of paying benefits.[3] In fact, Sharondale, which appeared through counsel, has not argued that it is incapable of paying benefits. Further, there is no showing of manifest injustice in dismissing Loftis Coal as the responsible operator, as there is a responsible operator for this claim. See *Director, OWCP v. Oglebay Norton Co.*, 877 F.2d 1300 (6th Cir. 1989)(liability should fall to Black Lung Disability Trust Fund only in absence of responsible operator, and not simply when a responsible operator was inefficiently reached). Additionally, the Director has not cited any authority that would permit it to indefinitely join Loftis as a party, just in case Sharondale cannot pay benefits sometime in the future. Therefore, I herein affirm the prior dismissal of Loftis Coal. It is not the responsible operator for this claim.

As to Sharondale, I find that it has been properly named as the responsible operator. Although it was not a party to the initial claim proceedings, Sharondale has had a full

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[2] See *Cox v. Benefits Review Bd.*, 791 F.2d 445, 447 (6th Cir. 1986) (court refused to consider argument petitioner failed to raise before Benefits Review Board); *Hix v. Director, OWCP*, 824 F.2d 526, 527 (6th Cir. 1986) ("the claimant may not obtain review of the ALJ's decision on any issue not properly raised before the Board").

[3] The record is devoid of any evidence that the Kentucky Coal Producers Self-Insurance Fund is bankrupt.

opportunity to defend the claim. Sharondale has not alleged nor specified any prejudice to it due to its late joinder. For example, there is no showing of any x-rays, etc., being unavailable to it for evaluation by its own physicians. Therefore, I find that Sharondale is the responsible operator for the payment of any benefits in this claim.

Medical Evidence

The following is a summary of the evidence submitted with the instant request for modification, some of it further re-readings of x-rays already in evidence:

A. Chest X-rays

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications[4] | Interpretation |
|---|---|---|---|---|
| DX 178 | 10/28/85 | 1 | Westerfield/B | Scarring at right diaphragm. 0/0. |
| DX 178 | 10/28/85 | 1 | West/B, BCR | Negative chest. 0/0. |
| DX 178 | 10/28/85 | 1 | Kendall/B, BCR | Negative chest. 0/0. |
| DX 178 | 10/28/85 | 1 | Poulos/B, BCR | Negative chest. 0/0. |
| DX 178 | 10/28/85 | 1 | Halbert/B, BCR | 0/0. Some scarring in right base associated with the right diaphragm. |
| DX 178 | 4/3/86 | 3 | Westerfield/B | Scarring at right base. 0/0. |
| DX 178 | 4/3/86 | 1 | West/B, BCR | Negative chest. 0/0. |
| DX 178 | 4/3/86 | 1 | Kendall/B, BCR | Negative chest. 0/0. |
| DX 178 | 4/3/86 | 2 | Poulos/B, BCR | Negative chest. 0/0. |
| DX 178 | 4/3/86 | 2 | Halbert/B, BCR | Negative chest. 0/0. |

[4] The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc. or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2).

The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a physician who has demonstrated expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. § 37.51 (1982).

| | | | | |
|---|---|---|---|---|
| DX 178 | 4/29/86 | 2 | Westerfield/B | Scarring at right mid diaphragm. 0/0. |
| DX 178 | 4/29/86 | 2 | West/B, BCR | Negative chest. 0/0. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 178 | 4/29/86 | 1 | Kendall/B, BCR | Negative chest. 0/0. |
| DX 178 | 4/29/86 | 2 | Poulos/B, BCR | Negative chest. 0/0. |
| DX 178 | 4/29/86 | 2 | Halbert/B, BCR | Negative chest. 0/0. |
| DX 146 and | 4/24/92 | - | Bofill (Hosp.) | Interstitial change suggestion of COPD. Right hemidiaphragm eventration. |
| DX 146 with right Flattened | 2/21/94 | - | Kim (Hospital) | Mild chronic diffuse interstitial change COPD. Tenting of hemidiaphragm. diaphragm. |
| DX 146 density width looks retro- sug- | 9/22/94 | - | Kim (Hospital) | Flattened diaphragm. Triangle-shaped in right lower chest without significant Change from 2/15/94, could be tenting of hemidiaphragm. Slightly increased size in of base from 1992; benign. Increased sternal space, may gest COPD. |

| | | | |
|---|---|---|---|
| DX 146 10/18/95 | - | Stebner (Hosp.) | Tenting of the right hemidiaphragm or scarring at the lung base. Nonspecific linear nodular pattern in both lungs with prominence of the hilar shadows, flattening of the diaphragms, and ill-defined soft tissue density in LLL. Chronic changes with COPD similar to 9-22-94. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 146 | 11/16/95 | - | Stebner (Hosp.) | Diffuse interstitial linear nodular pattern in both lung fields compatible with some type of pneumoconiosis. COPD. Tenting of right hemidiaphragm. |
| DX 144 | 10/29/97 | 1 | Rubenstein/B, BCR | 1/0, q/t, all six zones. Pleural thickening. |
| DX 145 | 10/29/97 | 2 | Bassali/B, BCR | ½, q/t, all six zones. Right non-calcified diaphragmatic pleural plaque. Kerley B-lines in both lung bases. |
| DX 147 | 10/29/97 | 2 | Sargent/B, BCR | 0/1, s, mid and lower zones, no evidence of CWP. Smoking history? Deformity right diaphragm, unknown etiology. |
| DX 148 | 10/29/97 | 2 | Barrett/B, BCR | Scarring RUL probably all secondary to prior inflammation. Bullae. Emphysema. Co. 0/0. |
| DX 178 | 10/29/97 | 2 | Westerfield/B | Generalized increase in bronchial markings suggests chronic bronchitis. Scarring at right mid diaphragm. 0/0. |
| DX 178 | 10/29/97 | 2 | West/B, BCR | Suspect mild COPD with increased pulmonary |

| | | | |
|---|---|---|---|
| | | | arterial pressures. 0/0. |
| DX 178 10/29/97 | 1 | Kendall/B, BCR | 0/0. Changes consistent with COPD. |
| DX 178 10/29/97 | 3 | Poulos/B, BCR | Negative chest. 0/0. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 178 right | 10/29/97 | 2 | Halbert/B, BCR | 0/0. Scarring in base associated with right diaphragm. |
| DX 168 and Pleural | 3/2/98 | 1 | Sundaram | 1/1, p/q, upper zones mid right zone. thickening. |
| DX 170 | 3/2/98 | 3 | Sargent/B, BCR | 0/0. Smoking history? Lungs hyperinflated. Calcified aortic arch. Localized lung volume loss right base. |
| DX 173 Active? | 3/1/99 | 3 | Sargent/B, BCR | Bullae? Emphysema? Tuberculosis? - Smoking history? Calcified aortic arch. Deformity right diaphragm? Etiology? LUL infiltrate, unknown etiology - active TB? |
| DX 178, mid DX 169 | 3/1/99 | 1 | Sundaram | 1/1, p/q, upper and zones. Pleural thickening and calcification. LUL scar. |
| DX 178 chronic tuberrespon- | 3/1/99 | 1 | West/B, BCR | LUL infiltrate with bullae, may be or active, an atypical pneumonia such as culosis could be sible. Suggestive |

| | | | | |
|---|---|---|---|---|
| | | | | underlying COPD. 0/0. |
| DX 178 | 3/1/99 | 1 | Kendall/B, BCR | LUL infiltrate. 0/0. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 178 | 3/1/99 | - | Hall (Hospital) | Left upper lobe and superior pneumonitis which may represent tuberculosis, fungal infection or atypical pneumonia. Neoplasm cannot be excluded. The appearance favors tuberculosis. |
| DX 178 | 3/1/99 | 1 | Poulos/B, BCR | LUL infiltrate which may be (2 readings) acute or chronic in nature.<br><br>Bullae changes LUL and apex. 0/0.<br><br>Underlying granulomatous disease process in LUL, such as tuberculosis, a should be consideration. |
| DX 178 | 3/1/99 | 2 | West/B, BCR | Bullous emphysema with bullae left apex. LUL infiltrate is worrisome for tuberculosis. Small nodular densities in both mid to lower lungs should be followed to exclude pulmonary nodules. 0/0.<br><br>Note: Resolved on later films. |
| DX 178 | 3/1/99 | 1 | Halbert/B, BCR | Large infiltrate LUL. Large bullae, left apex. |

| | | | | |
|---|---|---|---|---|
| | | | | Some scarring right base associated with right diaphragm. Due to scar-ring in left lung, evaluation for pneumoconiosis based on right lung - 0/0. |
| DX 178 | 3/1/99 | 1 | Kendall/B, BCR | 0/0. LUL interstitial infiltrate. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 178 | 5/26/99 | 1 | Patel/B, BCR | 1/0, t/s, 6 zones. Mild COPD with bilateral, upper zone bullous changes. Parenchymal scarring in LUL assoc- iated with disorganiza- tion of the pulmonary architecture. |
| DX 178 | 5/26/99 | 1 | Poulos/B, BCR | Bullae left paex. 0/0. |
| DX 178 | 5/26/99 | 1 | West/B, BCR | COPD with left apex bullae and interstitial scarring. Possible left significant pulmonary nodule. 0/0. |
| DX 178 | 5/26/99 | 1 | Halbert/B, BCR | 0/0. Because of the extensive scarring in left lung apex, evaluation for pneumoconiosis is based on the appearance of the right lung. Bullae. Di. |
| DX 178 | 5/26/99 | 1 | Kendall/B, BCR | 0/0. LUL interstitial infiltrate. |
| DX 178 | 6/23/99 | - | Hall (Hospital) | Atelectasis is most likely in RUL, cannot exclude pneumonia however. Scarring and retraction in LUL with bullous emphysematous changes, cannot entirely exclude a neoplasm or indolent process. |

| | | | | |
|---|---|---|---|---|
| DX 178 | 12/8/99 | 1 | Fino/B | Completely negative. |
| DX 178 | 12/8/99 | 1 | Poulos/B, BCR 0/0. | Bullae left apex. |

| Ex.No. | Date of X-ray | Film Qual. | Physician/ Qualifications | Interpretation |
|---|---|---|---|---|
| DX 178 | 12/8/99 | 1 | West/B, BCR | COPD with apical bullae and scarring. Possible neoplasm in LML. 0/0. |
| DX 178 | 12/8/99 | 2 | Halbert/B, BCR | 0/0 (right lung). Bullae. Di. |
| DX 178 | 12/8/99 | 1 | Kendall/B, BCR | 0/0. LUL interstitial infiltrate. |
| DX 178 | 12/18/99 | 1 | Dahhan/B | 0/0. Emphysema. |
| DX 178 | 12/18/99 | 3 | Sargent/B, BCR | 0/0. Bullae. Emphysema. Smoking history? Pulmo- nary arterial hyper- tension? Eventuation or herniation right hemi- diaphragm? |

B. CT Scans

The claimant underwent a CT scan on September 27, 1994 while hospitalized. Dr. J.H. Kim's impression was: "Previously seen triangle density in the right lower chest appears to be tenting right hemidiaphragm with fact. No definite mass is seen." (DX 146).

The claimant underwent another CT scan while he was hospitalized on June 27, 1999. The impression by Dr. Dan Hall was:

> Calcified, less than 1 cm, lesion in the left upper lobe likely representing a granuloma. Neoplasm is much less likely. A small focus of airspace disease is present in the right middle lobe posteriorly and likely represents atelectasis. The lesion in the left upper lobe could be followed by serial chest x-rays to verify stability.

(DX 178).

C. Pulmonary Function Studies

| Date | Ex. No. | Age/Hgt. | FEV1 | FVC | FEV1/ FVC | MVV | Coop/ Comp. |
|---|---|---|---|---|---|---|---|
| 6/2/86 | DX 160 | 43/66" | 1.18 | 2.78 | 42.45% | - - | - - |
| | | | * 1.72 | 3.49 | 49.28% | | |

Dr. Nausherwan K. Burki, who is board-certified in internal and pulmonary medicine, found the above study to be valid. (DX 160).

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 3/21/88 | DX 161 | 45/66" | 1.18 | 2.65 | 44.53% | - - | - - |
| | | | * 1.39 | 2.69 | 51.67% | | |
| 3/7/94 | DX 162 | 51/66" | 0.73 | 1.63 | 44.79% | - - | - - |
| | | | * 0.86 | 2.13 | 40.38% | | |
| 1/12/96 | DX 163 | 53/66" | 0.42 | 1.18 | 35.59% | - - | - - |
| | | | * 0.49 | 1.42 | 34.51% | | |

Dr. Burki also reviewed the latter three studies and determined that it was not acceptable due to the lack of original tracings. (DX 161, 162, 163). He also found the March 21, 1988 study invalid due to the variability in the curves indicating suboptimal effort. (DX 161).

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 5/26/99 | DX 178 | 56/64" | 0.91 | 3.46 | 26% | 39 | Good |
| | | | * 1.02 | 3.43 | 30% | 38 | |

* Results obtained post-bronchodilator.

D. Arterial Blood Gas Tests

| Date | Physician | pCO2 | pO2 | Ex. No. |
|---|---|---|---|---|
| 4/27/90 | Hospital | 36.3 | 74.3 | DX 146 |
| 4/24/92 | Hospital | 40.6 | 69.6 | DX 146 |
| 2/17/94 | Hospital | 71.7 | 60.6 | DX 152 |

Dr. Burki found the above study to be invalid as the PCO2 value was too high for the noted PO2 on room air. (DX 152).

| Date | Physician | pCO2 | pO2 | Ex. No. |
|---|---|---|---|---|
| 9/22/94 | Hospital | 39.5 | 42.9 | DX 153 |
| 9/23/94 | Hospital | 43.3 | 69.9 | DX 146 |
| 10/19/95 | Hospital | 50 | 50 | DX 154 |
| 11/16/95 | Hospital | 59.3 (on 2 liters of oxygen) | 60.5 | DX 155 |
| 11/17/95 | Hospital | 74 (on 4 liters of oxygen) | 75 | DX 156 |
| 11/12/96 | Sundaram | 58.4 | 63.8 | DX 157 |
| 11/16/96 | Sundaram | 57 | 60 | DX 158 |
| 12/13/97 | Sundaram | 47.7 | 60.3 | DX 159 |
| 5/26/99 | Rasmussen | 49.0<br>** 53.0 | 57.0<br>55.0 | DX 178 |
| 12/8/99 | Fino | 51.2 | 61.7 | DX 178 |
| 12/18/99 | Dahhan | 50.1<br>** 46.0 | 52.5<br>54.5 | DX 178 |

** Results obtained with exercise.

E. Hospital Records, Biopsy Reports, and Medical Examinations

The claimant was hospitalized at Williamson Memorial Hospital from April 24 to 27, 1992 due to severe headache, blurred vision, shortness of breath and high blood pressure. Previous admissions were for acute asthmatic bronchitis. Dr. Maximo Tan attended to the claimant, and rendered discharge

diagnoses of severe headache due to migraine; hypertension, uncontrolled; chronic obstructive pulmonary disease ("COPD"); and low back pain. (DX 146).

The claimant was next hospitalized at Williamson from February 16 to 20, 1994 due to bronchopneumonia. The discharge diagnoses set forth by Dr. Tan were bilateral interstitial pneumonia, COPD, and history of hypertension. (DX 146).

The claimant was readmitted from September 22 to 27, 1994 because of "severe shortness of breath, coughing and wheezing which he has been having for the past week and this has not been getting any better in spite of antibiotics that he has been taking at home." The medical history noted was prior "admissions to this hospital for the same problem of acute asthmatic bronchitis as well as pneumonia. The patient is a cigarette smoker in spite of advice to stop smoking." Dr. Tan was again the attending physician. The discharge diagnoses were acute asthmatic bronchitis, severe COPD, hypertension, and arthritis. (DX 146).

The claimant was next hospitalized at Williamson from October 18 to 21, 1995. The attending physician was Dr. Rosario Nadorra. A chest x-ray showed chronic interstitial changes consistent with emphysema with no acute infiltrate identified. The discharge diagnoses were acute respiratory failure secondary to COPD with acute exacerbation, acute on chronic low back strain, and hypertension. (DX 146).

The claimant was readmitted from November 16 to 21, 1995. The admitting impression was COPD with acute exacerbation, rule out respiratory failure. Dr. Tan was again the attending physician. A chest x-ray showed evidence of COPD and some interstitial lung disease. The discharge diagnoses were acute, severe bronchitis, with bronchospasm; advanced COPD; and severe leukocytosis, due to infections. (DX 146).

The claimant was next admitted to Williamson from November 12 to 16, 1996. Dr. Maan Younes was the attending physician. The discharge diagnoses were chronic obstructive pulmonary disease with acute exacerbation, hypertension, chronic low back pain, and anxiety disorder. (DX 146).

The claimant was hospitalized at Highlands Regional Medical Center from March 1 to 7, 1999 due to increasing shortness of breath, chest congestion, and respiratory distress without any improvement following medication. The attending physician was Dr. Raghu Sundaram. History included COPD and coal workers' pneumoconiosis, with no family history of tuberculosis. An
x-ray showed left upper lobe and superior segment pneumonitis and it was felt that tuberculosis needed to be ruled out. AFB initial smears were negative. The discharge diagnoses were bronchopneumonia with respiratory distress, chronic

obstructive pulmonary disease with exacerbation, coal workers' pneumoconiosis, arteriosclerotic heart disease, and rule out tuberculosis pending AFB cultures. (DX 175, 178).

On April 28, 1999, Dr. Sundaram wrote that:

> This gentleman has been seen by me for several years, his first visit was on 01-31-96 and the most recent was a follow-up visit on 12-19-98. His chief complaint is a history of shortness of breath on limited activity, smothering at night time. He has a history of smoking, he continues to smoke approximately ten (10) cigarettes a day. He also has a long history of coal exposure. ...
>
> Mr. Taylor cannot walk a distance of one block or go up flight of steps. He cannot lift any weight beyond ten pounds or carry the same over a few feet. The prognosis for Mr. Taylor would be in my professional opinion 1. coal workers pneumoconiosis; 2. chronic obstructive pulmonary disease. His problems definitely lays [sic] from his long exposure to coal dust of 21 ½ years. Considering his physical and significant impaired status, pulmonary function studies, x-rays, and blood gases that I have received from Williamson Hospital, he would be unable to indulge in any gainful employment and as such he is permanently and totally disabled. He is 56 years of age, again Mr. Taylor's disability would be due to his underlying condition of coal workers pneumoconiosis.
>
> Patient is advised to continue his oxygen on 24 hour basis and multiple medications he is on and follow up at the office as needed.

(DX 168, 169, 178).

Dr. D.L. Rasmussen interviewed and examined the claimant on May 26, 1999. The smoking history was 1 ½ packs of cigarettes per day from age eighteen in 1961; currently ½ pack per day. Family history included a father with asthma, emphysema, and black lung. Examination revealed low diaphragms, increased percussion note, moderately to markedly reduced breath sounds, inspiratory and expiratory wheezing and

rhonchi, and marked prolongation of the expiratory phase with forced respirations. An x-ray was read by Dr. Patel as positive for pneumoconiosis, 1/0. An electrocardiogram revealed sinus rhythm with moderate premature atrial contractions and occasional premature ventricular contractions and P pulmonale. A pulmonary function study showed severe, irreversible obstructive insufficiency. An arterial blood gas test at rest was abnormal, with marked hypoxia and moderate hypercarbia during exercise. Dr. Rasmussen concluded that:

> These studies indicate very severe, totally disabling respiratory insufficiency with evidence of probable cor pulmonale and pulmonary hypertension as reflected by the early anaerobic threshold. Obviously this patient would be totally disabled for resuming his last regular coal mine job.
>
> The patient has a significant history of exposure to coal mine dust. He has x-ray changes consistent with pneumoconiosis. It is medically reasonable to conclude that he has coalworkers' pneumoconiosis which arose from his coal mine employment.
>
> There appear to be 3 risk factors for this patient's disabling respiratory insufficiency. He does have a history suggestive of hyperactive airways disease, which, in fact could make him more vulnerable to the adverse effects of both cigarette smoking and coal mine dust exposure, the other two risk factors for his impairment. His coal mine dust exposure must be considered a significant contributing factor to his totally disabling respiratory insufficiency.

(DX 178).

The claimant was hospitalized again from June 23 to 30, 1999 due to recurrent episodes of vomiting, dehydration, shortness of breath, extreme weakness, and tightness. The attending physician was Dr. Sundaram. History included positive PPD with previous hospitalizations with no evidence for active tuberculosis on the AFB smears and cultures; COPD; coal workers' pneumoconiosis; and moderate anxiety. A chest x-ray showed atelectasis in the right middle lobe, the possibility of pneumonia considered, and bullous emphysematous

changes in the left upper lobe. A bronchoscopy was obtained, with biopsies of the left upper and right middle lobes. The specimens consisted of benign bronchial epithelium and pulmonary parenchyma, with the right lung showing a slight increase in anthracotic pigment beneath the bronchial mucosa. A CT scan was also obtained. The discharge diagnoses were bronchopneumonia, acute gastritis with dehydration, chronic obstructive pulmonary disease, coal workers' pneumoconiosis, and hypokalemia resolved with therapy. (DX 178).

On December 1, 1999, Dr. Sundaram wrote that:

> I have been treating Mr. Larry Taylor for several years now for shortness of breath due to COPD and Black Lung Disease. He has undergone many tests in the past and also recently, which have put much strain on his body. His condition is so severe that he should not undergo any more testing due to the stress that it creates on his body.

(DX 178).

Dr. B.T. Westerfield reviewed the medical records on behalf of the employer and issued a report on December 2, 1999. He concluded that, based on his x-ray readings and the majority of negative readings, the claimant does not have coal workers' pneumoconiosis. He concluded, however, that the claimant was totally disabled from pulmonary disease. He described it as "severe Chronic Obstructive Pulmonary Disease with both severe reduction in flow rates on spirometry and hypoxemia (low oxygen) with hypercarbia (elevated $CO_2$) on arterial blood gas," and related it to cigarette smoking. He stated that he did "not find any evidence that respiratory impairment in Mr. Taylor is related to Coal Workers' Pneumoconiosis." (DX 178).

On December 7, 1999, Dr. P. Raphael Caffrey reviewed the biopsy report of Dr. Braswell (hospital, June 30, 1999) at the employer's request, and stated that:

> The criteria for a pathologist to make a diagnosis of CWP was spelled out in the "Pathology

> Standards for Coal Worker's Pneumoconiosis" published in the Archives of Pathology and Laboratory Medicine, July 1979. Anthracotic pigment alone is not synonymous with CWP. The lesion of simple CWP consists of anthracotic pigment plus reticulin and usually focal emphysema.

Dr. Caffrey is board-certified in anatomical and clinical pathology. (DX 178).

Dr. Gregory J. Fino, who is board-certified in internal and pulmonary medicine, interviewed and examined the claimant on December 8, 1999 at the request of the employer. Examination of the chest revealed an increased AP diameter with a prolongation of the expiratory phase and wheezes on a forced expiration. An x-ray was interpreted as negative for pneumoconiosis. A pulmonary function study was not obtained due to the claimant's
treating physician's advice. An arterial blood gas test revealed moderate hypoxia and moderate hypercarbia. Dr. Fino also reviewed additional medical records. He concluded that the claimant was totally disabled due to severe chronic obstructive pulmonary disease due to smoking. He concluded that the claim-ant did not have an occupationally acquired pulmonary condition as a result of coal mine dust exposure because:

> 1. The majority of chest x-ray readings are negative for pneumoconiosis.
>
> 2. My reading of the chest x-ray is negative for pneumoconiosis.
>
> 3. The spirometric evaluations that have been performed show an obstructive ventilatory abnormality based on the reduction in the FEV1/FVC ratio. This obstructive ventilatory abnormality has occurred in the absence of any interstitial abnormality. In addition, the obstruction shows involvement in the small airways. Large airway flow is measured by the FEV1 and FEV1/FVC ratio. Small airway flow is measured by the FEF 25-75. On a proportional basis, the small airway flow is more reduced than the large airway flow. This type of finding is not consistent with a coal dust related condition but is consistent with conditions such as cigarette smoking, pulmonary

emphysema, non-occupational chronic bronchitis, and asthma. Minimal obstructive lung disease has been described in working coal miners and has been called industrial bronchitis. This condition is characterized by cough and mucous production plus minimal decreases in the FEV1 in some miners. Industrial bronchitis resolves within six months of leaving the mines. Obstructive lung disease may also arise from coal workers' pneumoconiosis when significant fibrosis is present. The fibrosis results in the obstruction. In this case, although obstruction can be seen in coal workers' pneumoconiosis, the obstruction is unrelated to coal mine dust exposure.

4. There is significant hypercarbia. This is consistent with smoking; it is not consistent with clinical or legal pneumoconiosis.

Dr. Fino added that:

> Even if industrial bronchitis due to coal mine employment contributed to the obstruction, the loss in FEV1 would be in the 200 cc range. If we gave back to him that amount of FEV1, this man would still be disabled. This medical estimate of loss in FEV1 in working miners was summarized in the 1995 NIOSH document. Although a statistical drop in the FEV1 was noted in working miners, that drop was not clinically significant. This man would be as disabled had he never stepped foot in the mines.

(DX 178).

Dr. Fino performed a record review on December 29, 1999. His conclusions remained the same. Additionally, Dr. Fino summarized and commented on the medical literature. The studies showed the following average losses in FEV1 in the noted countries: 108cc (UK), not significant (USA), 65cc (UK), 147cc (USA), 146cc (UK), 450cc (UK), 196cc (UK), no effect (USA), 113cc (USA), 495cc (USA), 1.8cc-531cc (USA), 2536cc (France), 108cc (USA), and 1440cc (Italy). Dr. Fino stated that:

> As an initial matter, the effect on FEV1 needs to be defined. All of the estimates noted above ... represent average losses of FEV1 assuming 45 years of working underground in the mines with a dust concentration of 2 mgm/$m^3$. This was calculated in order to compare and contrast the various studies. An average loss of FEV1 means that 50% of the miners will have losses in excess of the average and 50% will have losses smaller than the average. When applying this to an individual miner, one might as well flip a coin to make the decision whether the loss is greater than, or less than, the average. In other words, these articles merely reflect the law of probability, not statistical analysis or clinically significant findings.
>
> In addition, all of the studies that measured an average FEV1 loss are flawed because of selection bias. The results cannot be generalized to all miners. All of the authors discuss the problems

with selection bias and the limitations of the study. ...

Later in his report, Dr. Fino stated that:

> The studies which attempted to show a decrease in the FEV1 due to coal mine dust inhalation did not carefully control for, or consider, other potential risk
> factors for the decline in FEV1 apart from the usual factors such as aging, smoking and dust exposure measurements.
>
> Banks (3) noted that there is a "statistically significant relationship between mean FEV1 decline and dust exposure." He refers to a number of "other" potential factors for the decline in the FEV1 aside from smoking, age and dust:
>
> 1. Host susceptibility factors
> 2. Familial history of atopy
> 3. Childhood illnesses
> 4. Obesity and excessive weight gain
> 5. Intercurrent respiratory infection
> 6. Mine effect
> 7. Environment exposures, and
> 8. Socioeconomic status
>
> He goes on to state "attributing this effect to dust alone in any individual worker may not be reasonable unless specific information regarding the overall health of each worker is available. An assessment of the individual is necessary to understand the relationship between dust exposure, lung function decline and other medical problems."

Dr. Fino additionally stated that:

> There is no doubt that some miners do have clinically significant obstruction as a result of coal mine dust inhalation. This actually is expected in most cases of severe fibrosis where a combined obstructive and restrictive defect is present. However, there is no evidence that there is a clinically significant reduction in the FEV1 as a result of chronic obstructive lung disease due to coal mine
> dust inhalation. None of the studies show that.

The doctor further stated:

> The pathological description of coal workers' pneumoconiosis includes an entity called focal emphysema associated with the lesion of coal workers' pneumoconiosis. Some feel that this is centriacinar emphysema. The issue, however, is whether or not simple coal workers' pneumoconiosis or coal mine dust inhalation alone causes <u>clinically significant</u> emphysema. Whether or not it is referred to as focal or centriacinar is moot. The presence of emphysema in the lungs does not automatically imply respiratory impairment. The following does not pertain to complicated pneumoconiosis. It is well known that this condition may result in clinically significant emphysema and respiratory impairment.
>
> A review of the literature provides the following conclusions:
>
> 1. There has been confusion in the literature regarding the distinction between

> focal emphysema and centrilobular emphysema since both affect the same portion of the lung acinus. However, regardless of this debate, clinical impairment as a result of emphysema is the gold standard when evaluating a miner's pulmonary status.
>
> 2. The amount of emphysema in the lungs of miners increases with the severity of simple coal workers' pneumoconiosis. However, this is not true in simple silicosis.
>
> 3. Increasing severity of simple coal workers' pneumoconiosis (by radiograph or autopsy) is not correlated with a worsening of lung function.

As to particular studies, Dr. Fino commented that:

Dr. Wright and others published a "State of the Art" review on "Diseases of the Small Airways" (17). He discussed the association of mineral dusts and emphysema and commented that emphysema (pathologic) has been described in coal workers' pneumoconiosis. "The

lesions in coal workers have been termed focal emphysema. They appear as enlarged air spaces in the central portion of the lobule, and they bear a considerable resemblance to centrilobular emphysema induced by cigarette smoke, albeit the lesions in coal workers never appear to achieve the same severity as may be seen with smoke."

Dr. Gordon L. Snider also published a state-of-the-art review on emphysema (20, 21). He acknowledged that emphysema is a condition of the lung characterized by "enlargement of the respiratory air spaces" and described a number of different types of air space enlargement. In proximal acinar emphysema, the emphysema or enlargement of the air spaces begins in the respiratory bronchioles. He identifies two forms of proximal acinar emphysema. The first form is the "focal emphysema of simple coal workers' pneumoco-

> niosis" and the second form is "centrilobular emphysema". He distinguishes the centrilobular emphysema by stating that it is the "dominant form of emphysema in smokers."

(DX 178).

Dr. Abdul K. Dahhan interviewed and examined the claimant on December 18, 1999 at the request of the employer. The smoking history was two packs of cigarettes per day beginning at age twenty, cutting back to one-half pack per day three years ago
and quitting altogether three months ago. Examination of the chest revealed an increased AP diameter with hyperresonancy to percussion. Peripheral cyanosis was noted. An electrocardiogram showed regular sinus rhythm with a pattern of left anterior hemiblock. A pulmonary function study was declined on doctor's advice. An arterial blood gas test was obtained at rest and
with exercise, and the carboxyhemoglobin level was 6.4%. An x-ray was interpreted as negative for pneumoconiosis. Dr. Dahhan concluded that:

> 1. There is insufficient objective data to justify the diagnosis of coal workers' pneumoconiosis based on the obstructive abnormalities on clinical examination of the chest, the treatment program according to Mr. Taylor's family physician and negative x-ray reading for pneumoconiosis.
>
> 2. Mr. Taylor has advanced chronic obstructive lung disease of the variety of chronic bronchitis and emphysema.
>
> 3. Due to Mr. Taylor's decline of the pulmonary function studies, direct measurement of his true ventilatory capacity is not possible. However, I do not believe that he retains the respiratory capacity to return to his previous coal mining work or job of comparable physical demand.
>
> 4. Mr. Taylor's pulmonary disability did not result from coal dust exposure or occupational pneumoconiosis. He has not had any exposure to coal dust since 1985, a duration of absence sufficient to cause cessation of any industrial bronchitis that he

> may have had. Also, his family physician is treating him with multiple bronchodilators, including steroids and anti-asthma medication indicating that he believes that his condition is responsive to such therapy. These findings are inconsistent with the permanent adverse affects of coal dust on the respiratory system.
>
> 5. Mr. Taylor's obstructive airway disease has resulted from his 60+ pack years of smoking, an amount sufficient to cause the development of a disabling obstructive ventilatory defect in a susceptible individual. His carboxyhemoglobin level when I examined him was consistent with an individual smoking two packs per day, contradicting his statement that he had stopped smoking.
>
> 6. Mr. Taylor's pulmonary disability was not a result of coal dust exposure or coal workers' pneumoconiosis and I conclude that it would have developed at the same time and in the same manner regardless of whether or not he had ever worked in the coal mining industry or was exposed to coal dust.
>
> 7. Mr. Taylor has low back pain, essential hypertension and anxiety with depression. All are conditions of the general public at large and are not caused by, contributed to or aggravated by coal dust exposure or coal workers' pneumoconiosis.

Dr. Dahhan is board-certified in internal and pulmonary medicine. (DX 178).

Dr. Fino was deposed on April 12, 2000. He reiterated his findings. As to the difference in x-ray readings, Dr. Fino simply said that he disagreed with the positive readings. (DX 178).

Dr. Ben V. Branscomb, who is board-certified in internal medicine, reviewed the medical evidence on behalf of the employer and issued a report on June 26, 2000. As to the x-rays and CT scans, Dr. Branscomb stated that:

> Nearly everyone commented on the tenting or scarring at the right diaphragm beginning in 1985.

> Toward the more recent dates there were descriptions of pneumonias which then improved or resolved. One such pneumonia resulted in a left upper lobe scar.
>
> There is an overwhelming preponderance of negative readings for pneumoconiosis, including the opinions of many highly experienced "B" readers. Non-specific changes or COPD were noted by some persons. There were two CT scans. In neither of these were changes identified suggesting CWP. Dr. Stebner described non-specific linear and nodular changes and (sic) his interpretation of the CT of 09/27/94 or 09/28/94. His conclusion was that these were changes of COPD.

Dr. Branscomb's conclusion was that:

> There is no evidence of pneumoconiosis.
>
> Mr. Taylor was totally disabled to perform hard labor including coal mining. This was the result of chronic asthmatic bronchitis. This in turn was caused by a very severe smoking addiction plus a history of severe asthma and a positive family history of asthma and allergies. All his pulmonary problems were conditions of the general public and neither caused nor in any way aggravated or adversely influenced by coal dust exposure. He has no disability arising from his occupation as a coal miner with the exception of low back injuries.
>
> If I assume that Mr. Taylor has simple pneumoconiosis it would still be my conclusion that such pneumoconiosis neither caused his disabling obstructive
>
> pulmonary disease nor in any way aggravated or contributed to it.

(EX 1).

Dr. Branscomb was deposed on September 26, 2000. He testified that:

> It is well known that the combination of smoking in a person who has asthma is the most important predisposing risk factor for the production of chronic obstructive pulmonary disease. There's a name for that. It's called the Dutch Hypotheses because in the Netherlands they first realized that since everybody with asthma does not become totally disabled, who does? The answer is those people who both smoke and have asthma are much more likely to become disabled.
>
> The ongoing clinical course of the pulmonary disease in this gentleman was one of attacks of wheezing, attacks that produced acute sudden and severe worsening of the breathing. That is the pattern of asthma. When persons who have asthma either have it for a long time and fairly severely, and certainly if they smoked, they often have pronounced bronchitic symptoms as well. Now that justifies calling the diagnosis asthmatic bronchitis or bronchitis with asthma rather than simply pure asthma.

(EX 2).

Modification

Section 725.310 provides that a claimant may file a petition for modification within one year of the last denial of benefits. Modification petitions may be based upon a change in condition or a mistake in a determination of fact. 20 C.F.R. § 725.310(a).

In deciding whether the claimant has established a change in conditions, I must "perform an independent assessment of the newly submitted evidence, in conjunction with evidence previously submitted, to determine if the weight of the new evidence is sufficient to establish the element or elements which defeated entitlement . . .." Napier v. Director, OWCP, 17 BLR 1-111, 1-113 (1993). *See also* Nataloni v. Director, OWCP, 17 BLR 1-82, 1-84 (1993).

In deciding whether the prior decision contains a mistake in a determination of fact, I must review all the evidence of record, including evidence submitted since the most recent denial. New evidence, however, is not a prerequisite to modification based upon a mistake of fact. Nataloni, 17 BLR at 1-84; Kovac v. BCNR Mining Corp., 14 BLR 1-156, 1-158 (1990), *aff'd on recon.* 16 BLR 1-71, 1-73 (1992). *See also* O'Keefe v. Aerojet-General Shipyards, 404 U.S. 254, 257 (1971). Rather, the factfinder is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." O'Keefe, 404 U.S. at 257.

Because the claimant filed his application for benefits after March 31, 1980, this claim shall be adjudicated under the regulations at 20 C.F.R. Part 718. Under this part of the regulations, claimant must establish by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. Failure to establish any of these elements precludes entitlement to benefits. *See Anderson v. Valley Camp of Utah, Inc.*, 12 BLR 1-111, 1-112 (1989).

The medical evidence clearly shows that the claimant is totally disabled from a pulmonary/respiratory standpoint. Not one physician opined that the claimant is capable of resuming his former coal mine employment. Therefore, I modify the prior decision to find that the claimant is totally disabled. The remaining issues are thus whether the claimant has coal workers' pneumoconiosis, and whether his total disability is due to pneumoconiosis.

Pneumoconiosis and Causation

Under the Act, pneumoconiosis is defined as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b). Section 718.202(a) provides four methods for determining the existence of pneumoconiosis: X-ray evidence, biopsy or autopsy evidence, application of a presumption, and medical opinion evidence. 20 C.F.R. §§ 718.202(a)(1)-(4).

I find no error in the previous judge's determination that the x-ray evidence does not establish pneumoconiosis. At best, the evidence is in equipoise: although the majority of readings

are negative, a few dually-qualified physicians have read x-rays as positive, thereby showing that the x-rays can be read either way. However, such does not establish pneumoconiosis under § 718.202(a)(1).

The biopsy evidence is insufficient for a determination of pneumoconiosis. A finding of anthracotic pigmentation is not equivalent to a finding of pneumoconiosis. § 718.202(a)(2);
report of Dr. Caffrey. Therefore, I find that pneumoconiosis is not established under § 718.202(a)(2). However, "[a] negative biopsy is not conclusive evidence that the miner does not have pneumoconiosis." § 718.106(c).

None of the referenced presumptions are applicable. Therefore, pneumoconiosis cannot be established under §718.202(a)(3).

The record contains numerous medical opinions. With the exception of Dr. Sundaram, who appears to have overlooked the matter, all of the physicians agreed that smoking either contributed to the claimant's impairment (COPD [chronic bronchitis/ asthmatic bronchitis/emphysema]) or totally caused it. As to the contribution of coal dust exposure, there are several physicians who opined that it did not contribute at all. Other physicians, namely Drs. Mettu, Penman, and Rasmussen, opined that both coal dust exposure and cigarette smoking have to be considered as causes of the claimant's impairment. Dr. Sundaram related the COPD entirely to coal workers' pneumoconiosis.

The statutory definition of pneumoconiosis includes "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." § 718.201.

Dr. Mettu's opinion was obtained on referral from the OWCP, pursuant to § 725.405(a). The previous judge found Dr. Mettu's opinion to be equivocal on the cause of the claimant's impairment, and his finding was upheld on appeal. Dr. Mettu

stated that the claimant's impairment could be due to smoking or coal dust exposure; he did not provide any reasoning. I also note that Dr. Mettu's qualifications were not submitted into evidence. Given the case law that provides that the Director has not properly discharged his duty under § 725.405(a) if the physician's opinion is incomplete, *Pettry v. Director, OWCP*, 14 BLR 1-98 (1990); *Hall v. Director, OWCP,* 14 BLR 1-51 (1990), and the Director did not provide a new examination when this request for modification was filed, I am remanding the matter for the Director to fulfill its obligation under § 725.405(a). That an opinion has to be documented and reasoned is set forth at § 718.202(a)(4).

Clearly, a physician needs to address the scientific evidence in this record in order to provide a well-reasoned opinion. Dr. Fino's review of the medical literature lends credence to those physicians who state that they cannot distinguish the impairment from coal dust from the impairment of smoking when an individual has a history of both. Dr. Fino's report shows that focal and centriacinar/centrilobular emphysema develop in the same area of the lung. His report indicates to me that the dispute is not what type of emphysema is deemed to be present, but whether one believes that coal dust can make a clinically significant contribution to the development of emphysema. His interpretation of the data is that it does not.

Drs. Branscomb and Rasmussen emphasized the history of asthma, and Dr. Fino also specified other factors that can influence the development of COPD. Thus, a well-reasoned opinion needs to address these factors, although the Act does not limit benefits because a miner has an increased susceptibility to the development of occupational lung disease. I note that in addition to a family history of asthma, the claimant gave a history that his father had emphysema and black lung.

Therefore, based on the foregoing, I am herein remanding the claim to the OWCP for a § 725.406(a) examination, which may be limited given the claimant's poor health. However, the physician shall be provided with the medical record so that he or she can render a documented opinion. The physician's credentials are to be submitted as well. The parties may submit additional evidence following that report.

ORDER

IT IS ORDERED THAT:

Loftis Coal Company, Inc. is dismissed from this claim.

The claim of Larry Taylor for benefits under the Act is remanded to the District Director, Office of Workers' Compensation Programs, for a § 725.406(a) examination and medical report.

A
JOSEPH E. KANE
Administrative Law Judge